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#5	Search **dvl-3 or (dishevelled-3) or (dishevelled 3) and (cancer or tumor or carcinoma or malignancy) and (inhibitor or antagonist)** Limits: **Publication Date to 2003/7/31**	13:23:40	4
#8	Search **(#2 and expression)** Limits: **Publication Date to 2003/7/31**	13:20:57	13
#20	Search **(dvl 3) or (dvl-3) and (cancer or carcinoma or tumor or tumour or malignancy)** Limits: **Publication Date to 2003/7/31**	13:19:38	9
#7	Search **X2 and expression** Limits: **Publication Date to 2003/7/31**	12:49:59	184
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DATE: Friday, April 20, 2007

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☐	L4	(xu)[IN] AND (dishevelled)	7
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       20apr07 15:28:00 User290558 Session D103.1
            $0.97    0.278 DialUnits File1
     $0.97  Estimated cost File1
     $0.14  INTERNET
     $1.11  Estimated cost this search
     $1.11  Estimated total session cost   0.278 DialUnits

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S (DVL (W) 3) OR (DISHEVELLED)
             725  DVL
        13077535  3
               41  DVL(W)3
             1798  DISHEVELLED
      S1     1806  (DVL (W) 3) OR (DISHEVELLED)
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S S1 AND (CANCER OR CARCINOMA OR TUMOR OR TUMUOR OR MALIGNANCY)
             1806  S1
          3683078  CANCER
          1827555  CARCINOMA
          3461327  TUMOR
               58  TUMUOR
           247831  MALIGNANCY
      S2      332  S1 AND (CANCER OR CARCINOMA OR TUMOR OR TUMUOR OR
                   MALIGNANCY)
?

S S2 AND (INHIBITOR? OR ANTAGONIST?)
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          3281480  INHIBITOR?
          1387386  ANTAGONIST?
      S3       84  S2 AND (INHIBITOR? OR ANTAGONIST?)
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S S3 AND SIRNA
               84  S3
            21094  SIRNA
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RD S4
      S5        2  RD S4  (unique items)
?

TYPE S5/FULL/1-2

  5/9/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14892738 PMID: 15150100

Inhibition of Wnt-1 signaling induces apoptosis in beta-catenin-deficient mesothelioma cells.

You Liang; He Biao; Uematsu Kazutsugu; Xu Zhidong; Mazieres Julien; Lee Amie; McCormick Frank; Jablons David M

Thoracic Oncology Laboratory, Department of Surgery, Comprehensive Cancer Center, University of California, San Francisco, California 94115, USA.

Cancer research (United States) May 15 2004, 64 (10) p3474-8, ISSN 0008-5472--Print Journal Code: 2984705R

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

It is known that Wnt-1 signaling inhibits apoptosis by activating beta-catenin/tcf-mediated transcription. Here, we show that blocking Wnt-1 signaling in beta-catenin-deficient mesothelioma cell lines H28 and MS-1 induces apoptotic cell death. Both Wnt-1 small interfering RNA (siRNA) and Dishevelled siRNA induced significant apoptosis in these cell lines. A small molecule inhibitor of c-Jun NH(2)-terminal kinase inhibited the apoptotic cell killing induced by either Wnt-1 siRNA or Dishevelled siRNA in these cells. Our data suggest that beta-catenin-independent noncanonical pathway(s), i.e., Wnt/JNK pathway, may play a role in the apoptotic inhibition caused by Wnt-1 signaling.

Descriptors: *Apoptosis--physiology--PH; *Cytoskeletal Proteins --deficiency--DF; *Mesothelioma--pathology--PA; *Proto-Oncogene Proteins --antagonists and inhibitors--AI; *Trans-Activators--deficiency--DF; Carcinoma, Non-Small-Cell Lung--genetics--GE; Carcinoma, Non-Small-Cell Lung--pathology--PA; Cytoskeletal Proteins--genetics--GE; Cytoskeletal Proteins--physiology--PH; Humans; Lung Neoplasms--genetics--GE; Lung Neoplasms--pathology--PA; Mesothelioma--genetics--GE; Proto-Oncogene Proteins--physiology--PH; RNA, Small Interfering--administration and dosage --AD; RNA, Small Interfering--genetics--GE; Research Support, Non-U.S. Gov't; Signal Transduction--physiology--PH; Trans-Activators--genetics--GE; Trans-Activators--physiology--PH; Transfection; Wnt Proteins; Wnt1 Protein ; beta Catenin

CAS Registry No.: 0 (CTNNB1 protein, human); 0 (Cytoskeletal Proteins) ; 0 (Proto-Oncogene Proteins); 0 (RNA, Small Interfering); 0 (Trans-Activators); 0 (WNT1 protein, human); 0 (Wnt Proteins); 0 (Wnt1 Protein); 0 (beta Catenin)

Record Date Created: 20040519

Record Date Completed: 20040802

5/9/2 (Item 1 from file: 5)

18191819 BIOSIS NO.: 200500097732

Dishevelled promotes neurite outgrowth in neuronal differentiating neuroblastoma 2A cells, via a DIX-domain dependent pathway

AUTHOR: Fan Shongshan; Ramirez Servio H; Garcia Tatiana M; Dewhurst Stephen (Reprint)

AUTHOR ADDRESS: Ctr MedDept Microbiol and Immunol, Univ Rochester, 601 Elmwood Ave,Box 672, Rochester, NY, 14652, USA**USA

AUTHOR E-MAIL ADDRESS: stephendewhurst@urmc.rochester.edu

JOURNAL: Molecular Brain Research 132 (1): p38-50 December 6, 2004 2004

MEDIUM: print

ISSN: 0169-328X _(ISSN print)

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Dishevelled (Dvl) is a cytoplasmic protein involved in the Writ-Frizzled signaling cascade, which has also been shown to interact with the cytoskeleton in part through inhibition of glycogen synthase kinase 3beta (GSK3beta). Using mouse neuroblastoma 2A (N2A) cells as a model system, we have found that overexpression of Dvl promotes the outgrowth of neurite-like processes, and leads to the induction of a striking, bipolar morphologic phenotype during neuronal differentiation. In contrast, suppression of Dvl expression using isoform-specific siRNAs led to an inhibition of neurite outgrowth in these cells. In order to further elucidate the mechanism(s) responsible for this effect, we overexpressed several mutant forms of Dvl in the N2A cells, including deletions in each of the three major functional subdomains of the protein (DELTADIX, DELTAPDZ, DELTADEP) and point mutations in the two well-defined interaction motifs within the DIX domain (the actin-binding and vesicle-association elements; K58A and K68A/E69A, respectively). These experiments revealed that the DIX domain (and its vesicle-binding subregion) was essential for Dvl's effect on neurite extension and morphogenesis in N2A cells. In contrast, direct overexpression of a degradation-resistant form of beta-catenin (S37A), or a dominant negative GSK3beta mutant (K85R), had no effect on neurite outgrowth or morphology in neuronally differentiating N2A cells; exposure of cells to a pharmacologic inhibitor of GSK3 (lithium) also had no effect. Taken together, these results suggest that Dvl induces cytoskeletal and morphologic rearrangements in neuronal differentiating N2A cells through a mechanism that cannot be attributed exclusively to modulation of GSK3beta or beta-catenin activity, but which does depend upon a DIX-domain/vesicle-association-dependent signaling pathway.

REGISTRY NUMBERS: 443900-95-6: glycogen synthase kinase 3 beta
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Molecular Genetics --Biochemistry and Molecular Biophysics; Nervous System--Neural Coordination; Tumor Biology
BIOSYSTEMATIC NAMES: Adenoviridae--dsDNA Viruses, Viruses, Microorganisms ; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Adenovirus (Adenoviridae)--gene vector; N2A cell line (Muridae)--morphogenesis
ORGANISMS: PARTS ETC: neuron--nervous system, differentiation, outgrowth
COMMON TAXONOMIC TERMS: Double-Stranded DNA Viruses; Microorganisms; Viruses; Animals; Chordates; Mammals; Nonhuman Vertebrates; Nonhuman Mammals; Rodents; Vertebrates
DISEASES: neuroblastoma--neoplastic disease, nervous system disease
MESH TERMS: Neuroblastoma (MeSH)
CHEMICALS & BIOCHEMICALS: beta-catenin--activity; dishevelled--expression; glycogen synthase kinase 3 beta--activity; siRNA
GENE NAME: N2A cell line GSK3beta gene (Muridae)--mutant
MISCELLANEOUS TERMS: neuritogenesis
CONCEPT CODES:
02506 Cytology - Animal
03502 Genetics - General
03506 Genetics - Animal
10060 Biochemistry studies - General
20504 Nervous system - Physiology and biochemistry
20506 Nervous system - Pathology
24004 Neoplasms - Pathology, clinical aspects and systemic effects

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  31500 Genetics of bacteria and viruses
  33502 Virology - General and methods
BIOSYSTEMATIC CODES:
  03116 Adenoviridae
  86375 Muridae
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Set     Items   Description
S1       1806   (DVL (W) 3) OR (DISHEVELLED)
S2        332   S1 AND (CANCER OR CARCINOMA OR TUMOR OR TUMUOR OR MALIGNAN-
           CY)
S3         84   S2 AND (INHIBITOR? OR ANTAGONIST?)
S4          5   S3 AND SIRNA
S5          2   RD S4  (unique items)
?

S S2 AND SIRNA
             332  S2
           21094  SIRNA
      S6      18  S2 AND SIRNA
?

RD S6
      S7       8  RD S6  (unique items)
?

TYPE S7/FULL/1-7

  7/9/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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20176129   PMID: 15913453
 Efficacy of Wnt-1 monoclonal antibody in sarcoma cells.
  Mikami Iwao; You Liang; He Biao; Xu Zhidong; Batra Sonny; Lee Amie Y;
Mazieres Julien; Reguart Noemi; Uematsu Kazutsugu; Koizumi Kiyoshi; Jablons
David M
  Department of Surgery, Comprehensive Cancer Center, University of
California, San Francisco, CA 94115, USA. imikami@cc.ucsf.edu
  BMC cancer electronic resource (England)     2005, 5 (1) p53, ISSN
1471-2407--Electronic   Journal Code: 100967800
  Publishing Model Electronic
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  Subfile:   INDEX MEDICUS
  BACKGROUND: Sarcomas are one of the most refractory diseases among
malignant tumors. More effective therapies based on an increased
understanding of the molecular biology of sarcomas are needed as current
forms of therapy remain inadequate. Recently, it has been reported that
Wnt-1/beta-catenin signaling inhibits apoptosis in several cancers. In this
study, we investigated the efficacy of a monoclonal anti-Wnt-1 antibody in
sarcoma cells. METHODS: We treated cell lines A-204, SJSA-1, and fresh
primary cultures of lung metastasis of sarcoma with a monoclonal anti-Wnt-1
antibody. Wnt-1 siRNA treatment was carried out in A-204. We assessed cell
death using Crystal Violet staining. Apoptosis induction was estimated by
flow cytometry analysis (Annexin V and PI staining). Cell signaling changes
were determined by western blotting analysis. RESULTS: We detected Wnt-1
```

expression in all tissue samples and cell lines. Significant apoptosis induction was found in monoclonal anti-Wnt-1 antibody treated cells compared to control monoclonal antibody treated cells ($p < 0.02$). Similarly, we observed increased apoptosis in Wnt-1 siRNA treated cells. Blockade of Wnt-1 signaling in both experiments was confirmed by analyzing intracellular levels of Dishevelled-3 and of cytosolic beta-catenin. Furthermore, the monoclonal anti-Wnt-1 antibody also induced cell death in fresh primary cultures of metastatic sarcoma in which Wnt-1 signaling was active. CONCLUSION: Our results indicate that Wnt-1 blockade by either monoclonal antibody or siRNA induces cell death in sarcoma cells. These data suggest that Wnt-1 may be a novel therapeutic target for the treatment of a subset of sarcoma cells in which Wnt-1/beta-catenin signaling is active.

Descriptors: *Antibodies, Monoclonal--therapeutic use--TU; *Lung Neoplasms--secondary--SC; *Lung Neoplasms--therapy--TH; *Sarcoma--therapy --TH; *Wnt1 Protein--immunology--IM; Annexin A5--pharmacology--PD; Antibodies, Monoclonal--chemistry--CH; Apoptosis; Blotting, Western; Cell Line, Tumor; Flow Cytometry; Fluorescent Dyes--pharmacology--PD; Gentian Violet--pharmacology--PD; Humans; Lung Neoplasms--immunology--IM; Neoplasm Metastasis; Propidium--pharmacology--PD; Proteins--metabolism--ME; RNA Interference; RNA, Small Interfering--metabolism--ME; Research Support, Non-U.S. Gov't; Sarcoma--embryology--EM; Sarcoma--immunology--IM; Sarcoma --pathology--PA; Signal Transduction; Tumor Cells, Cultured; Wnt1 Protein --chemistry--CH; Wnt1 Protein--physiology--PH; beta Catenin--metabolism --ME

CAS Registry No.: 0 (Annexin A5); 0 (Antibodies, Monoclonal); 0 (DVL3 protein, human); 0 (Fluorescent Dyes); 0 (Proteins); 0 (RNA, Small Interfering); 0 (Wnt1 Protein); 0 (beta Catenin); 36015-30-2 (Propidium); 548-62-9 (Gentian Violet)

Record Date Created: 20050629

Record Date Completed: 20060327

Date of Electronic Publication: 20050524

7/9/2 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

15575433 PMID: 16007226

Inhibition of Wnt16 in human acute lymphoblastoid leukemia cells containing the t(1;19) translocation induces apoptosis.

Mazieres Julien; You Liang; He Biao; Xu Zhidong; Lee Amie Y; Mikami Iwao; McCormick Frank; Jablons David M

UCSF Comprehensive Cancer Center, San Francisco, CA 94115, USA.

Oncogene (England) Aug 11 2005, 24 (34) p5396-400, ISSN 0950-9232--Print Journal Code: 8711562

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

The Wnt family of secreted glycoproteins is widely involved in cell proliferation, differentiation and oncogenesis. Many Wnt signaling genes are upregulated and activated in chronic lymphocytic leukemia. Less is known concerning acute leukemia. One subtype of acute lymphoblastoid leukemia (ALL) is characterized by a t(1;19) chromosomal translocation resulting in a fusion protein E2A-Pbx1 that promotes transformation and leukemogenesis. Wnt16 has been shown to be targeted by E2A-Pbx1. We performed a differential gene expression array in acute leukemia cell lines

displaying or not displaying the t(1;19) translocation. We found that Wnt16 and many Wnt signaling-related genes were upregulated in the translocation-containing cells. As two isoforms of Wnt16, Wnt16a and Wnt16b, have been recently identified, we demonstrated by using RT-PCR and Western blot that Wnt16b (and not Wnt16a) is overexpressed in t(1;19)-containing cell lines. We then directly addressed the role played by both isoforms in this type of leukemia. Using specific short interfering RNA (siRNA) and an anti-Wnt16 antibody, we showed that targeted-Wnt16b inhibition leads to apoptotic cell death. We also demonstrated that Wnt16b mediates its effect through the canonical Wnt pathway involving dishevelled-2, beta-catenin and survivin. We thus propose that Wnt16 plays an important role in leukemogenesis, raising its therapeutic interest.

Descriptors: *Apoptosis--genetics--GE; *Chromosomes, Human, Pair 1; *Chromosomes, Human, Pair 19; *Glycoproteins--physiology--PH; *Leukemia, Lymphocytic, Acute--genetics--GE; *Leukemia, Lymphocytic, Acute--pathology --PA; *Translocation, Genetic; Gene Expression Profiling; Glycoproteins --biosynthesis--BI; Glycoproteins--genetics--GE; Humans; RNA, Small Interfering; Research Support, Non-U.S. Gov't; Reverse Transcriptase Polymerase Chain Reaction; Tumor Cells, Cultured; Up-Regulation; Wnt Proteins

CAS Registry No.: 0 (Glycoproteins); 0 (RNA, Small Interfering); 0 (WNT16 protein, human); 0 (Wnt Proteins)

Record Date Created: 20050812

Record Date Completed: 20050901

7/9/3 (Item 3 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

14892738 PMID: 15150100

Inhibition of Wnt-1 signaling induces apoptosis in beta-catenin-deficient mesothelioma cells.

You Liang; He Biao; Uematsu Kazutsugu; Xu Zhidong; Mazieres Julien; Lee Amie; McCormick Frank; Jablons David M

Thoracic Oncology Laboratory, Department of Surgery, Comprehensive Cancer Center, University of California, San Francisco, California 94115, USA.

Cancer research (United States) May 15 2004, 64 (10) p3474-8, ISSN 0008-5472--Print Journal Code: 2984705R

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

It is known that Wnt-1 signaling inhibits apoptosis by activating beta-catenin/tcf-mediated transcription. Here, we show that blocking Wnt-1 signaling in beta-catenin-deficient mesothelioma cell lines H28 and MS-1 induces apoptotic cell death. Both Wnt-1 small interfering RNA (siRNA) and Dishevelled siRNA induced significant apoptosis in these cell lines. A small molecule inhibitor of c-Jun NH(2)-terminal kinase inhibited the apoptotic cell killing induced by either Wnt-1 siRNA or Dishevelled siRNA in these cells. Our data suggest that beta-catenin-independent noncanonical pathway(s), i.e., Wnt/JNK pathway, may play a role in the apoptotic inhibition caused by Wnt-1 signaling.

Descriptors: *Apoptosis--physiology--PH; *Cytoskeletal Proteins --deficiency--DF; *Mesothelioma--pathology--PA; *Proto-Oncogene Proteins --antagonists and inhibitors--AI; *Trans-Activators--deficiency--DF; Carcinoma, Non-Small-Cell Lung--genetics--GE; Carcinoma, Non-Small-Cell Lung--pathology--PA; Cytoskeletal Proteins--genetics--GE; Cytoskeletal

Proteins--physiology--PH; Humans; Lung Neoplasms--genetics--GE; Lung Neoplasms--pathology--PA; Mesothelioma--genetics--GE; Proto-Oncogene Proteins--physiology--PH; RNA, Small Interfering--administration and dosage --AD; RNA, Small Interfering--genetics--GE; Research Support, Non-U.S. Gov't; Signal Transduction--physiology--PH; Trans-Activators--genetics--GE; Trans-Activators--physiology--PH; Transfection; Wnt Proteins; Wnt1 Protein ; beta Catenin

CAS Registry No.: 0 (CTNNB1 protein, human); 0 (Cytoskeletal Proteins) ; 0 (Proto-Oncogene Proteins); 0 (RNA, Small Interfering); 0 (Trans-Activators); 0 (WNT1 protein, human); 0 (Wnt Proteins); 0 (Wnt1 Protein); 0 (beta Catenin)

Record Date Created: 20040519

Record Date Completed: 20040802

7/9/4 (Item 4 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

14537633 PMID: 14562050

Activation of the Wnt pathway in non small cell lung cancer: evidence of dishevelled overexpression.

Uematsu Kazutsugu; He Biao; You Liang; Xu Zhidong; McCormick Frank; Jablons David Mark

Thoracic Oncology Laboratory, UCSF Cancer Center, University of California at San Francisco, San Francisco, CA 94115, USA.

Oncogene (England) Oct 16 2003, 22 (46) p7218-21, ISSN 0950-9232--Print Journal Code: 8711562

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Non small cell lung cancer (NSCLC) is the leading cause of cancer deaths in the United States and worldwide. Unfortunately, standard therapies remain inadequate. An increased understanding of the molecular biology of lung cancer biology is required to develop more effective new therapies. In this report, we show that the Wnt pathway is activated through Dishevelled (Dvl) overexpression in NSCLC. Analysis of freshly resected tumors and lung cancer cell lines demonstrate that Dvl-3, a critical mediator of Wnt signaling, is overexpressed. Specifically, Dvl-3 was overexpressed significantly in 75% of fresh NSCLC microdissected samples compared to control paired matched normal lung samples. To evaluate the biological significance of Wnt signaling and, in particular, Dvl function in lung cancer, we transfected siRNA (designed to inhibit selectively human Dvl-1, -2, and -3), to the NSCLC cell line H1703, which is known to have beta-catenin-mediated Tcf-dependent transcriptional activity. Here, we demonstrate that Dvl-specific siRNA treatment in H1703 decreases significantly Dvl and beta-catenin expression, resulting in reduction of Tcf-dependent transcriptional activity, and, importantly, growth inhibition. Taken together, these data support the novel hypothesis that Dvl overexpression is critical to Wnt signaling activation and cell growth in NSCLC.

Descriptors: *Carcinoma, Non-Small-Cell Lung--genetics--GE; *Gene Expression Regulation, Neoplastic--genetics--GE; *Lung Neoplasms--genetics --GE; *Proto-Oncogene Proteins--genetics--GE; *Zebrafish Proteins; Adenocarcinoma--genetics--GE; Carcinoma, Non-Small-Cell Lung--enzymology --EN; Carcinoma, Squamous Cell--genetics--GE; Humans; Lung Neoplasms --enzymology--EN; Protein-Tyrosine Kinase--genetics--GE; RNA, Small

Interfering--genetics--GE; Tumor Stem Cell Assay; Wnt Proteins
CAS Registry No.: 0 (Proto-Oncogene Proteins); 0 (RNA, Small Interfering); 0 (Wnt Proteins); 0 (Zebrafish Proteins); 0 (wnt8b protein, zebrafish)
Enzyme No.: EC 2.7.1.112 (Protein-Tyrosine Kinase)
Record Date Created: 20031016
Record Date Completed: 20031126

7/9/5 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18191819 BIOSIS NO.: 200500097732
Dishevelled promotes neurite outgrowth in neuronal differentiating neuroblastoma 2A cells, via a DIX-domain dependent pathway
AUTHOR: Fan Shongshan; Ramirez Servio H;. Garcia Tatiana M; Dewhurst Stephen (Reprint)
AUTHOR ADDRESS: Ctr MedDept Microbiol and Immunol, Univ Rochester, 601 Elmwood Ave,Box 672, Rochester, NY, 14652, USA**USA
AUTHOR E-MAIL ADDRESS: stephendewhurst@urmc.rochester.edu
JOURNAL: Molecular Brain Research 132 (1): p38-50 December 6, 2004 2004
MEDIUM: print
ISSN: 0169-328X _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Dishevelled (Dvl) is a cytoplasmic protein involved in the Writ-Frizzled signaling cascade, which has also been shown to interact with the cytoskeleton in part through inhibition of glycogen synthase kinase 3beta (GSK3beta). Using mouse neuroblastoma 2A (N2A) cells as a model system, we have found that overexpression of Dvl promotes the outgrowth of neurite-like processes, and leads to the induction of a striking, bipolar morphologic phenotype during neuronal differentiation. In contrast, suppression of Dvl expression using isoform-specific siRNAs led to an inhibition of neurite outgrowth in these cells. In order to further elucidate the mechanism(s) responsible for this effect, we overexpressed several mutant forms of Dvl in the N2A cells, including deletions in each of the three major functional subdomains of the protein (DELTADIX, DELTAPDZ, DELTADEP) and point mutations in the two well-defined interaction motifs within the DIX domain (the actin-binding and vesicle-association elements; K58A and K68A/E69A, respectively). These experiments revealed that the DIX domain (and its vesicle-binding subregion) was essential for Dvl's effect on neurite extension and morphogenesis in N2A cells. In contrast, direct overexpression of a degradation-resistant form of beta-catenin (S37A), or a dominant negative GSK3beta mutant (K85R), had no effect on neurite outgrowth or morphology in neuronally differentiating N2A cells; exposure of cells to a pharmacologic inhibitor of GSK3 (lithium) also had no effect. Taken together, these results suggest that Dvl induces cytoskeletal and morphologic rearrangements in neuronal differentiating N2A cells through a mechanism that cannot be attributed exclusively to modulation of GSK3beta or beta-catenin activity, but which does depend upon a DIX-domain/vesicle-association-dependent signaling pathway. Copyright 2004 Elsevier B.V. All rights reserved.

REGISTRY NUMBERS: 443900-95-6: glycogen synthase kinase 3 beta
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Molecular Genetics

--Biochemistry and Molecular Biophysics; Nervous System--Neural Coordination; Tumor Biology
BIOSYSTEMATIC NAMES: Adenoviridae--dsDNA Viruses, Viruses, Microorganisms ; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Adenovirus (Adenoviridae)--gene vector; N2A cell line (Muridae)--morphogenesis
ORGANISMS: PARTS ETC: neuron--nervous system, differentiation, outgrowth
COMMON TAXONOMIC TERMS: Double-Stranded DNA Viruses; Microorganisms; Viruses; Animals; Chordates; Mammals; Nonhuman Vertebrates; Nonhuman Mammals; Rodents; Vertebrates
DISEASES: neuroblastoma--neoplastic disease, nervous system disease
MESH TERMS: Neuroblastoma (MeSH)
CHEMICALS & BIOCHEMICALS: beta-catenin--activity; dishevelled--expression; glycogen synthase kinase 3 beta--activity; siRNA
GENE NAME: N2A cell line GSK3beta gene (Muridae)--mutant
MISCELLANEOUS TERMS: neuritogenesis
CONCEPT CODES:
02506 Cytology - Animal
03502 Genetics - General
03506 Genetics - Animal
10060 Biochemistry studies - General
20504 Nervous system - Physiology and biochemistry
20506 Nervous system - Pathology
24004 Neoplasms - Pathology, clinical aspects and systemic effects
31500 Genetics of bacteria and viruses
33502 Virology - General and methods
BIOSYSTEMATIC CODES:
03116 Adenoviridae
86375 Muridae

7/9/6 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17950389 BIOSIS NO.: 200400321146
Multiple mechanisms for Wnt11-mediated repression of the canonical Wnt signaling pathway
AUTHOR: Maye Peter; Zheng Jie; Li n; Wu Dianqing (Reprint)
AUTHOR ADDRESS: Ctr HlthDept Genet and Dev Biol, Univ Connecticut, MC3301,263 Farmington Ave, Farmington, CT, 06030, USA**USA
AUTHOR E-MAIL ADDRESS: dwu@neuron.uchc.edu
JOURNAL: Journal of Biological Chemistry 279 (23): p24659-24665 June 4, 2004 2004
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The effect of a noncanonical Wnt, Wnt11, on canonical Wnt signaling stimulated by Wnt1 and activated forms of LRP5 (low density lipoprotein receptor-related protein-5), Dishevelled1 (Dvl1), and beta-catenin was examined in NIH3T3 cells and P19 embryonic carcinoma cells. Wnt11 repressed Wnt1-mediated activation of LEF-1 reporter activity in both cell lines. However, Wnt11 was unable to inhibit canonical signaling activated by LRP5, Dvl1, or beta-catenin in NIH3T3 cells, although it could in P19 cells. In addition, Wnt11-mediated inhibition of canonical signaling in NIH3T3 cells is ligand-specific; Wnt11 could effectively repress canonical signaling activated by Wnt1,

Wnt3, or Wnt3a but not by Wnt7a or Wnt7b. Co-culture experiments with NIH3T3 cells showed that the co-expression of Wnt11 with Wnt1 was not an essential requirement for the inhibition, suggesting receptor competition as a possible mechanism. Moreover, in both cell types, elevation of intracellular Ca2+ levels, which can result from Wnt11 treatment, led to the inhibition of canonical signaling. This result suggests that Wnt11 might not be able to signal in NIH3T3. Furthermore, P19 cells were found to express both endogenous canonical Wnts and Wnt11. Knockdown of Wnt11 expression using siRNA resulted in increased LEF-1 reporter activity, thus indicating that Wnt11-mediated suppression of canonical signaling exists in vivo.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Cell Biology
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: NIH3T3 cell line (Muridae)--murine fibroblast cells; P19 cell line (Muridae)--murine embryonic carcinoma cells
COMMON TAXONOMIC TERMS: Animals; Chordates; Mammals; Nonhuman Vertebrates ; Nonhuman Mammals; Rodents; Vertebrates
CHEMICALS & BIOCHEMICALS: Dvl1 {Dishevelled 1}; Wnt1; Wnt11; Wnt3; Wnt3a; Wnt7a; beta-catenin; low-density lipoprotein; low-density lipoprotein receptor; low-density lipoprotein receptor-related protein-5
MISCELLANEOUS TERMS: canonical Wnt signaling pathway--multiple Wnt11-mediated repression mechanisms
CONCEPT CODES:
02502 Cytology - General
02506 Cytology - Animal
10060 Biochemistry studies - General
10064 Biochemistry studies - Proteins, peptides and amino acids
10066 Biochemistry studies - Lipids
BIOSYSTEMATIC CODES:
86375 Muridae

7/9/7 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

13390056 EMBASE No: 2005467061
Efficacy on Wnt-1 monoclonal antibody in sarcoma cells
Mikami I.; You L.; He B.; Xu Z.; Batra S.; Lee A.Y.; Mazieres J.; Reguart N.; Uematsu K.; Koizumi K.; Jablons D.M.
Dr. D.M. Jablons, Department of Surgery, Comprehensive Cancer Center, 1600 Divisadero St., San Francisco, CA 94115 United States
AUTHOR EMAIL: jablonsd@surgery.ucsf.edu
BMC Cancer (BMC CANCER) (United Kingdom) 24 MAY 2005, 5/- (7p)
CODEN: BCMAC ISSN: 1471-2407
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 21

Background: Sarcomas are one of the most refractory diseases among malignant tumors. More effective therapies based on an increased understanding of the molecular biology of sarcomas are needed as current forms of therapy remain inadequate. Recently, it has been reported that Wnt-1/beta-catenin signaling inhibits apoptosis in several cancers. In this study, we investigated the efficacy of a monoclonal anti-Wnt-1 antibody in sarcoma cells. Methods: We treated cell lines A-204, SJSA-1, and fresh

primary cultures of lung metastasis of sarcoma with a monoclonal anti-Wnt-1 antibody. Wnt-1 siRNA treatment was carried out in A-204. We assessed cell death using Crystal Violet staining. Apoptosis induction was estimated by flow cytometry analysis (Annexin V and PI staining). Cell signaling changes were determined by western blotting analysis. Results: We detected Wnt-1 expression in all tissue samples and cell lines. Significant apoptosis induction was found in monoclonal anti-Wnt-1 antibody treated cells compared to control monoclonal antibody treated cells ($p<0.02$). Similarly, we observed increased apoptosis in Wnt-1 siRNA treated cells. Blockade of Wnt-1 signaling in both experiments was confirmed by analyzing intracellular levels of Dishevelled-3 and of cytosolic beta-catenin. Furthermore, the monoclonal anti-Wnt-1 antibody also induced cell death in fresh primary cultures of metastatic sarcoma in which Wnt-1 signaling was active. Conclusions: Our results indicate that Wnt-1 blockade by either monoclonal antibody or siRNA induces cell death in sarcoma cells. These data suggest that Wnt-1 may be a novel therapeutic target for the treatment of a subset of sarcoma cells in which Wnt-1/beta-catenin signaling is active. (c) 2005 Mikami et al; licensee BioMed Central Ltd.

DRUG DESCRIPTORS:
*monoclonal antibody--pharmacology--pd
small interfering RNA; Wnt1 protein--endogenous compound--ec; beta catenin --endogenous compound--ec; unclassified drug
MEDICAL DESCRIPTORS:
*sarcoma cell
drug efficacy; cancer cell culture; lung metastasis; apoptosis; flow cytometry; Western blotting; protein expression; signal transduction; human ; controlled study; human tissue; human cell; article
DRUG TERMS (UNCONTROLLED): Wnt1 protein antibody--pharmacology--pd
SECTION HEADINGS:
  016 Cancer
  030 Clinical and Experimental Pharmacology
  037 Drug Literature Index
?

Set	Items	Description
S1	1806	(DVL (W) 3) OR (DISHEVELLED)
S2	332	S1 AND (CANCER OR CARCINOMA OR TUMOR OR TUMUOR OR MALIGNANCY)
S3	84	S2 AND (INHIBITOR? OR ANTAGONIST?)
S4	5	S3 AND SIRNA
S5	2	RD S4 (unique items)
S6	18	S2 AND SIRNA
S7	8	RD S6 (unique items)

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 NEWS 17  FEB 26   CAS Registry Number crossover limit increased from 10,000
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 NEWS 21  MAR 22   LWPI reloaded
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=> s (dvl-3) or dishevelled
L1         1059 (DVL-3) OR DISHEVELLED

=> s L1 and (antagonist or inhibitor)
L2          165 L1 AND (ANTAGONIST OR INHIBITOR)

=> s L2 and (cancer or tumor or tumuot or carcinoma or malignancy)
L3           62 L2 AND (CANCER OR TUMOR OR TUMUOT OR CARCINOMA OR MALIGNANCY)

=> s L3 and siRNA
L4            2 L3 AND SIRNA

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L5   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2007:113586  CAPLUS
DN   146:226597
TI   Gene expression profiles in esophageal cancer and their use in
     diagnosis, prognosis, therapy and drug design and selection
IN   Nakamura, Yusuke; Daigo, Yataro; Nakatsuru, Shuichi
PA   Oncotherapy Science, Inc., Japan; The University of Tokyo
SO   PCT Int. Appl., 249pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
PI   WO 2007013671        A2    20070201    WO 2006-JP315342        20060726
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP,
             KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN,
             MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU,
             SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG,
             US, UZ, VC, VN, ZA, ZM, ZW
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RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI US 2005-703263P P 20050727

AB In order to identify the mols. involved in esophageal carcinogenesis and those to be useful for diagnostic markers as well as targets for new drugs and immunotherapy, a cDNA microarray representing 32,256 genes was constructed to analyze the expression profiles of 19 esophageal squamous-cell carcinomas (ESCCS) purified by laser-capture microdissection. A detailed genome-wide database for sets of genes that are significantly up- or down-regulated in esophageal cancer is disclosed herein. These genes find use in the development of therapeutic drugs or immunotherapy as well as tumor markers. Addnl., genes associated with lymph-node metastasis and post-surgery recurrence are disclosed herein. Among the candidate mol. target genes, a Homo sapiens epithelial cell transforming sequence 2 oncogene (ECT2) and a cell division cycle 45, S. cerevisiae, homolog-like (CDC45L) are further characterized. Treatment of ESCC cells with small interfering RNAs (siRNAs) of ECT2 or CDC45L suppressed growth of the cancer cells. Thus, the data herein provide valuable information for identifying diagnostic systems and therapeutic target mols. for esophageal cancer. Furthermore, the present inventors have identified DKK1 as a potential biomarker for diagnosis of cancer such as lung and esophageal cancers as well as prediction of the poor prognosis of the patients with these diseases. DKK1 was specifically over-expressed in most lung and esophageal cancer tissues the present inventors examined, and was elevated in the sera of a large proportion of patients with these tumors. DKK1, combined with other tumor markers, could significantly improve the sensitivity of cancer diagnosis. Moreover, this mol. is also a likely candidate for development of therapeutic approaches such as antibody therapy.

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AN 2004136202 ESBIOBASE

TI Inhibition of Wnt-1 signaling induces apoptosis in β -catenin-deficient mesothelioma cells

AU You L.; He B.; Uematsu K.; Xu Z.; Mazieres J.; Lee A.; McCormick F.; Jablons D.M.

CS D.M. Jablons, Department of Surgery, Cancer Center, Box 1674, 1600 Divisadero Street, San Francisco, CA 94115, United States. E-mail: jablonsd@surgery.ucsf.edu

SO Cancer Research, (15 MAY 2004), 64/10 (3474-3478), 33 reference(s) CODEN: CNREA8 ISSN: 0008-5472

DT Journal; Article

CY United States

LA English

SL English

AB It is known that Wnt-1 signaling inhibits apoptosis by activating β-catenin/tcf-mediated transcription. Here, we show that blocking Wnt-1 signaling in β-catenin-deficient mesothelioma cell lines H28 and MS-1 induces apoptotic cell death. Both Wnt-1 small interfering RNA (siRNA) and Dishevelled siRNA induced significant apoptosis in these cell lines. A small molecule inhibitor of c-Jun NH.sub.2-terminal kinase inhibited the apoptotic cell killing induced by either Wnt-1 siRNA or Dishevelled siRNA in these cells. Our data suggest that β-catenin-independent noncanonical pathway(s), i.e., Wnt/JNK pathway, may play a role in the apoptotic inhibition caused by Wnt-1 signaling.

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☐	L9	L1 and siRNA	14
☐	L8	L4 and expression and siRNA and cancer	13
☐	L7	L4 and expression and siRNA	14
☐	L6	L4 and (lung and mesothelioma and breast) and expression	6
☐	L5	L4 and (lung and mesothelioma and breast)	6
☐	L4	L2 and (inhibitor or antagonist)	52
☐	L3	L2 and (inhibitor or antagnoist)	52
☐	L2	L1 and (cancer or tumor or carcinoma or malignancy)	54
☐	L1	(dvl-3) or (dvl3)	57

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